Weight bearing asymmetry

Weight bearing asymmetry in standing hemiparetic patients

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MCA stroke may disrupt corticobulbar projection to brainstem output pathways involved in the vestibular control of balance

Postural disorders are a primary disability after stroke.[1] They lead to loss of autonomy and expose patients to a high risk of falling. We must bear in mind that following a total anterior circulation infarct, the median time to recover the ability to stand for 10 s is 44 days (25th–75th percentile: 38–57 days)[2]. Retraining the patient to stand is therefore a primary goal in post stroke rehabilitation, especially following hemisphere strokes.

Three main patterns characterise the standing posture of hemiparetic patients[1]: i) an increase in centre of gravity displacement, which reflects postural instability and results from orthopaedic, sensorimotor, and cognitive impairments; ii) the presence of a small area of stability beyond which the centre of gravity cannot move without exposing the patient to a loss of balance (this results either from an inability to control a stressed equilibrium system or from impaired co-ordination between posture and movement); and iii) weight bearing asymmetry, with more weight on the non-paretic leg. Unstable standing posture, although a major target in stroke rehabilitation, is still poorly understood. Weakness certainly plays a role, as do cognitive disorders observed in patients with lesions of the right hemisphere.[3] These cognitive disorders result in distortion of the coordinates used to distribute loading over the two legs while standing. Since some patients align their erect posture to a biological vertical contralesionally tilted, it has also been suggested that the shift in the center of gravity towards the ipsilesional leg might be a compensatory strategy to prevent contralesional falling.[1]

In a very elegant experimental study published in this issue (***pp 670–8***), Marsden *et al* propose a new approach to the problem of postural instability in standing hemiparetic patients. Using the measurement of forces and movements elicited by galvanic and transcranial electrical stimulation, they have explored the possibility of asymmetric vestibulo-spinal excitability in chronic middle cerebral artery (MCA) stroke patients, in patients with isolated corticospinal tract lesions, and in normal subjects. Patients and subjects were required to stand barefoot on two separate force plates with equal weight on both legs and with eyes closed. One advantageous feature of galvanic vestibular stimulation is that it is possible to evoke a bilateral response by stimulating vestibular afferents on one side only,[4 5] which means any asymmetries that may exist in the response pattern can be identified. It also offers an opportunity to decide whether asymmetry arises from an abnormality in the processing of sensory information (altered response in both legs for a lateralised deficit of sensory information) or an abnormality in the motor control of one side of the body (altered response in one leg and not the other irrespective of which ear is stimulated). The main finding of the Marsden *et al* study was abnormal interleg response asymmetry to galvanic vestibular stimulation in stroke patients only, the amplitude of the response being higher on the non-paretic side than on the paretic side, and higher on the non-paretic side than in controls. Since the changes in the reaction forces were observed early after vestibular stimulation (320–500 ms), the authors assumed that this initial response was likely to be purely vestibular in origin. Marsden *et al* also found that the degree of asymmetry induced by galvanic stimulation was correlated with the degree of corticospinal damage induced by transcranial magnetic stimulation. They concluded that MCA stroke may disrupt corticobulbar projection to brainstem output pathways which are involved in the vestibular control of balance. They suggested that stroke is associated with a lateralised deficit in the motor output stage of vestibular processing rather than in the sensory or spatial processing stages.

Postural rehabilitation must be guided by a better understanding of the postural disorders displayed by patients. There is no doubt that the paper by Marsden *et al* contributes to this knowledge. This new insight on the postural instability of standing stroke patients must now be confirmed and integrated with knowledge of the many other alterations and deficiencies which are involved in postural disorders caused by hemisphere strokes.

J Neurol Neurosurg Psychiatry 2005;**76**:621. doi: 10.1136/jnnp.2004.050468

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Competing interests: none declared

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